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Little evidence for the usefulness of violence risk assessment reply

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The 'rest of medicine' and psychiatry: why paradigms would differ

In their paper, Bracken *et al*¹ have cogently put forth the limitations of psychiatry comparing its differences with the 'rest of medicine'. They turn our attention to some moral and ethical notions viz relationships, meanings and values, which not only have therapeutic scope but also humanistic importance. Applying evidence-based logic, they show the inadequacy of technological interventions (psychopharmacotherapeutics or therapy-specific aspects of psychotherapies), and at the same time cite evidence of effectiveness of 'non-technical' aspects of care. Considering some of these aspects and the online response it generated, it is important that we refocus our attention to a central and some associated issues.

First, unlike what Bracken *et al* propose, medicine's assumptions on causal mechanisms are still a hotly debated issue. Medicine's apparent authority over human health was convincingly questioned in a historical analysis by Thomas McKeown and his arguments much advanced by Simon Szreter. In short, rather than technical innovations in medicine (such as the advent of antibiotics or immunisation), social and political interventions had a decisive role in advancing human health.²

Second, as the field of epidemiology progressively advances and uses newer analytic techniques, monocausal explanations (as the germ theory of disease propounded) gave way to multicausal (as in the case of chronic disease epidemiology) and finally to complex eco-epidemiological causal explanations.³ In fact, an active engagement with the notion of embodiment that explains how biological processes are influenced profoundly by environmental determinants (e.g. social, cultural, economic, political) lies at the heart of social epidemiology.⁴ And biological outcomes are not often mediated by our psyche, although the latter may be similarly affected.

Third, an attempt to compare the effect sizes of pharmacological interventions in both general medical disorders and psychiatric disorders show, barring a few exceptions, that effect sizes of psychiatric drugs are in the same range (i.e. small to medium) as most other pharmacotherapeutics.⁵

Moreover, the oft referred crisis in psychiatry also bothers the 'rest of medicine' and healthcare. Some features of this crisis are

the increasing difficulty of grappling with the explosive boom in health-related technologies (consequently increasing the cost of healthcare), the challenge produced by the epidemiological shift in disease prevalence and the marked social inequalities in health. In addition, the notions of 'medicalisation of everyday life'/overmedicalisation, healthism, biomedicalisation and the dominance of the technological paradigm in medicine have also drawn wide criticism. In not considering these as entirely good or bad, the problem is the undue attention to individualised solutions and personalised/customised technologies,⁶ transforming health to individual moral responsibility.⁷

On the other hand, under the foregoing transformations in healthcare, medical training instils qualities such as objectivity and emotional distancing to maintain clinical neutrality, concepts partly counterposed to values, narratives and meanings. Similarly, clinicians have come to associate professional status and power with increasing technological involvement in clinical practice, rather than with being sensitive to the patient's distress and life story. Although clinical knowledge is based on biological understanding and scientific methods, it is also interpretive and narrative.⁸

Thus to paraphrase Bracken *et al*, it is not just mental health problems but all health problems in general that undoubtedly have a biological dimension, and that by their very nature can reach beyond the body to involve social, cultural and psychological dimensions.

- 1 Bracken P, Thomas P, Timimi S, Asen E, Behr G, Beuster C, et al. Psychiatry beyond the current paradigm. *Br J Psychiatry* 2012; **201**: 430–4.
- 2 Szreter S. Rethinking McKeown: the relationship between public health and social change. *Am J Public Health* 2002; **92**: 722–5.
- 3 Krieger N. Proximal, distal, and the politics of causation: what's level got to do with it? *Am J Public Health* 2008; **98**: 221–30.
- 4 Krieger N, Smith GD. 'Bodies count' and body counts: social epidemiology and embodying inequality. *Epidemiol Rev* 2004; **26**: 92–103.
- 5 Leucht S, Hierl S, Kissling W, Dold M, Davis JM. Putting the efficacy of psychiatric and general medicine medication into perspective: review of meta-analyses. *Br J Psychiatry* 2012; **200**: 97–106.
- 6 Crawford R. Healthism and the medicalization of everyday life. *Int J Health Serv* 1980; **10**: 365–88.
- 7 Clarke AE, Mamo L, Fishman JR, Shim JK, Fosket JR. Technoscientific transformations of health, illness, and U.S. biomedicine. *Am Sociol Rev* 2003; **68**: 161–94.
- 8 Montgomery K. *How Doctors Think: Clinical Judgment and the Practice of Medicine*. Oxford University Press, 2006.

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Authors' reply: We are broadly in agreement with the thrust of Dr Das's analysis. In our original article, we cited Arthur Kleinman's call for 'medicine in general' to go beyond a technicalised understanding of 'caregiving' and we also noted the resonance between our position and that of Iona Heath in relation to general practice.

We agree entirely that 'an active engagement with the notion of embodiment' would represent a very positive agenda for all of medicine. Our experiences as human beings are shaped by our physiology and the particular way it has evolved over centuries. However, they are also shaped by the particular cultural and historical context in which, and through which, we come to know ourselves and the world around us. In the lived reality of human beings, mind, body and social context are inseparable.

But a medicine that sees itself as, primarily, a set of technical interventions will always strive to compartmentalise and conceptualise illness in simplified causal models. This represents a challenge for all branches of medicine.

Are we wrong to distinguish psychiatry from the 'rest of medicine'? Maybe. Bill Fulford has argued convincingly that the widely held view that bodily illness is 'relatively transparent in meaning' and less 'value-laden' than mental illness does not stand up to scrutiny.¹ For him, it is simply that the values inherent in our concepts of bodily disorder are just not as obvious as those involved in our discourse of mental illness. When the presenting problem is pain from an arthritic joint or from a myocardial infarction, there is usually agreement between the doctor, the patient and the carer about what the priorities are and what would count as recovery. However, as medical technologies (such as in reproductive healthcare) develop, more areas of disagreement emerge and ethical issues become more obvious. In the world of mental health, disagreements about values, priorities and frameworks have always been part of day-to-day work and thus value judgements more obvious.

However, although we accept this analysis, we are not entirely satisfied that this is the full story. When we put the adjective 'mental' in front of the word 'illness', we do seem to be delineating a particular territory of human suffering. This cannot be clearly defined and seems to resist easy categorisation. But the word 'mental' implies that this is suffering that emerges from the mind, and whatever the 'mind' is, it is not simply another organ of the body. In this way, there does seem to be some sort of epistemological difference between psychiatry and other branches of medicine such as cardiology, endocrinology or neurology. Problems with our thoughts, feelings, behaviours and relationships would seem to be more intimately entwined with questions of meaning and context than problems arising from lesions in specific organs of the body.

Whatever we make of the relationship between bodily and mental illness, psychiatry grapples daily with epistemological and ontological issues and has a long history of doing so. A psychiatry that is able to 'move beyond the current paradigm' might be one that can offer insights and leadership to other parts of medicine.

1 Fulford KWM. *Moral Theory and Medical Practice*. Cambridge University Press, 1989.

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Low Apgar scores in neonates with prenatal antidepressant exposure

We read with interest the very important and thought-provoking study by Jensen *et al.*¹ The authors have found an increased rate of low Apgar scores in neonates with prenatal antidepressant exposure, especially with selective serotonin reuptake inhibitors (SSRIs).¹ However, the use of other antidepressants (new or old) and a diagnosis of maternal depression were not associated with low Apgar scores.¹ The study has several merits: nationwide data, large sample size, meticulous record keeping, sound methodology, appropriate use of statistics, controlling confounders to a large extent and, most importantly, having been conducted in a clinically relevant area, where data were limited and there were more questions than answers.

However, there are certain issues with the study. First, the authors have not mentioned which of the SSRIs was implicated in having the greatest or least effect on lowering Apgar score. Second, the dose and duration of antidepressant use were not mentioned and adherence to antidepressants was also not assessed. Third, antidepressant data were collected from psychiatric centres only, perhaps because the authors did not have access to data from general practitioners, which further limits the generalisability of the study findings. Fourth, the authors have not mentioned and not controlled for important confounders such as the presence of a physical disorder in the mother, obstetric complications and nutritional status of mothers, which may also contribute to a low Apgar score. Fifth, there is a possible mistake in tabulating the gestational age of all pregnancies, as the interquartile range is stated as 39–39 weeks (see Table 1). Finally, the authors have themselves mentioned about the significant differences in the antidepressant prescription trends. During the study period, use of antidepressants was very limited in pregnant women, but recently antidepressant use has increased substantially, especially that of SSRIs. This may be an important reason for getting high odds ratios for low Apgar scores with the use of an SSRI. Earlier studies have also reported low Apgar scores with maternal SSRI use.^{2,3} Exposure to SSRIs at an early age can disrupt the normal maturation of the serotonin system and alter serotonin-dependent neuronal processes in the fetus³ and these effects are partly moderated by infant SLC6A4 genotype.⁴

Today, authors have advised caution and proper monitoring of infants with prenatal antidepressant exposure. This study will definitely provide impetus for future research in this area, and with more robust data, it may also act as a starting point for the modification of existing treatment guidelines.

- 1 Jensen HM, Grøn R, Lidgaard Ø, Pedersen LH, Andersen PK, Kessing LV. Maternal depression, antidepressant use in pregnancy and Apgar scores in infants. *Br J Psychiatry* 2013; **202**: 347–51.
- 2 Lund N, Pedersen LH, Henriksen TB. Selective serotonin reuptake inhibitor exposure in utero and pregnancy outcomes. *Arch Pediatr Adolesc Med* 2009; **163**: 949–54.
- 3 Oberlander TF, Bonaguro RJ, Misri S, Papsdorf M, Ross CJ, Simpson EM. Infant serotonin transporter (SLC6A4) promoter genotype is associated with adverse neonatal outcomes after prenatal exposure to serotonin reuptake inhibitor medications. *Mol Psychiatry* 2008; **13**: 65–73.
- 4 Maciag D, Simpson KL, Coppinger D, Lu Y, Wang Y, Lin RC, et al. Neonatal antidepressant exposure has lasting effects on behavior and serotonin circuitry. *Neuropsychopharmacology* 2006; **31**: 47–57.

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Authors' reply: We would like to emphasise that our study included nationwide data on the use of all antidepressants in Denmark wherever prescribed (including from primary care), however nationwide data on the diagnosis of depression were only available from in-patient and out-patient psychiatric hospital settings (and not from primary care). Thus, as argued in our paper, we believe our findings can be generalised to all women taking antidepressants during pregnancy regardless of the indication for treatment (depression, anxiety disorder, etc.) or the severity of illness.

Although, the study included more than 34 000 women who used an antidepressant before or during pregnancy, this number was too small for separate analyses of the individual antidepressants divided into the eight risk groups defined in the study. Register-based medication studies at present do not have access to data on the dose of drug treatment or on patient adherence to the drug. We did try to adjust our analyses for physical disorder in the mother as all analyses were adjusted for all other types of medication (in addition to antidepressants) that the mother may have used during pregnancy, in this way taking account of treated physical and mental disorders as well as depressive and anxiety disorders. We further adjusted analyses for maternal age, employment status, smoking status, calendar year, parity, gender of the newborn, \pm birth weight and \pm gestational age, however we did not include data on nutrition of the mother and on obstetric complications as suggested. Obstetric complications may rather be intermediary factors than confounders.

Regarding the gestational age of all mothers, this was correctly indicated in Table 1 as a median of 39 (interquartiles 39–39), as infants with a gestational age less than 22 weeks were excluded from analyses and the vast majority of children were born within week 39.

Like Nebhinani & Soni, we hope the study will provide impetus for future research in this increasingly important area, especially as the use of antidepressants during pregnancy is believed to increase even further in the future.

Declaration of interest

L.V.K. has been a consultant for Bristol-Myers Squibb, Eli Lilly, Lundbeck, AstraZeneca, Pfizer, Wyeth, Servier, and Janssen-Cilag.

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intensities than those typically used to treat seasonal affective disorder' cannot be supported by the findings of this study, since there was not an adequate control group. Further, the authors acknowledge that a number of non-specific factors may account for any improvements in depression and anxiety and all participants received relaxation. I strongly suspect that the fact that the participants had their eyes open during relaxation does not negate the effects on anxiety that relaxation training might have. In addition, most of the improvement in both groups (particularly on the depression subscale) had occurred before they were exposed to the intervention, i.e. at T_2 .

The clinical trial registration indicates that the control arm should have been receiving 100 lux for 30 min a day and the active arm 10 000 lux for 30 min a day. The study suggests that both arms received 20 min of light per day, with the control arm receiving an intensity of 2000 lux. It is not clear why the intensity was increased.

The attrition rate was high in both groups: 18/45 (40%) in the control arm and 15/46 (32.6%) in the active arm. Five patients in the active arm had an increase in seizures or required their medication to be increased (compared with two patients in the control arm). In the other paper emerging from this study,² the authors caution about using bright light in this population because 'it may result in an increase in seizures for some'. None of this caution is evident in the paper published in the *British Journal of Psychiatry*. Indeed, there is not a single mention of adverse effects, despite them being reported elsewhere.

The analysis does not appear to have been intention-to-treat, and the results are only reported for those patients that completed the trial. This is a significant weakness when the authors have reported the possibility of adverse effects in other journals and when the attrition rates are relatively high. It is not clear why this intervention in an epilepsy population is treated with some reservations, yet it is reported much more favourably when there are some improvements in a secondary outcome measure reflecting some aspects of mental health (anxiety and depressive symptoms) which occurred before the intervention.

- 1 Baxendale S, O'Sullivan J, Heaney D. Bright light therapy for symptoms of anxiety and depression in focal epilepsy: randomised controlled trial. *Br J Psychiatry* 2013; **202**: 352–6.
- 2 Baxendale S, O'Sullivan J, Heaney D. Bright light therapy as an add on treatment for medically intractable epilepsy. *Epilepsy Behav* 2012; **24**: 359–64.

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Are the conclusions supported by the evidence?

Many people might be confused about the term 'placebo' that is used in Baxendale *et al*'s study.¹ The paper clearly refers to the low-intensity-light arm as receiving placebo treatment, and the clinical trial registration (<http://clinicaltrials.gov/show/NCT01028456>) also indicates that the low-intensity group is receiving a placebo. However, this has some implications for the interpretation of the results.

If the low-intensity arm is indeed a placebo, the active treatment group did not differentiate from placebo and this is, therefore, a negative study. If, however, the low-intensity arm is receiving an active treatment then there is no placebo group and we cannot determine whether any changes in symptoms were due to the treatment or would have occurred by chance.

The conclusions that light therapy may 'be an effective treatment for symptoms of low mood in epilepsy at lower

Author's reply: Dr Christmas is quite correct in reiterating the uncertainty we expressed in our discussion about the placebo condition in our study. This does indeed have very significant implications for the interpretation of our results. It is for this reason that we suggested a number of different interpretations for our findings in the Discussion, including the possibility that light therapy 'may, therefore, be an effective treatment for symptoms of low mood in epilepsy at lower intensities than those typically used to treat seasonal affective disorder'. We also discussed the possibility that this could indeed be a negative finding or that the results we found could be due to other factors unrelated to light therapy, such as the establishment of fixed morning routines.

Dr Christmas is correct in that in the original protocol for the study the control arm should have been receiving 100 lux. The modifications to the original protocol were submitted with the paper as an online appendix, to conform to the CONSORT

guidelines for reporting trials. In this case, when the light boxes were modified to 100 lux, the disparity in intensity was very obvious and we did not feel that the study would conform to the important double-blind aspect of the design. It would have been very clear to any patient who received the 100 lux box that they had been assigned to the low-intensity arm of the trial. We therefore modified the boxes to administer 2000 lux at 20 min in the low-intensity arm. The boxes appeared bright, but literature on seasonal affective disorder indicates that this would not be a therapeutic dose within this time frame, whereas 10 000 lux at 20 min would be a therapeutic intensity/dose.

As we stated in the introduction to our study, the primary outcome measure for this trial was seizure control. We have reported these results separately¹ and that paper is fully referenced in our study. Although it is possible that bright light therapy may result in an increase in seizures for some patients, this was not a statistically significant finding in our previous study and, as yet, the risk remains theoretical. Clinicians will be aware that seizure control should be carefully monitored following the introduction of any new treatment offered to people with epilepsy.

In presenting the results of our study for publication we have sought to provide as clear an account of the data as possible. The results are by no means clear-cut or definitive. However, there are some interesting aspects to the data that suggest that this may not be a dead end in terms of a treatment option for some people with epilepsy. This study stands as a guide for future research. We hope that its limitations, which we fully acknowledge and have set out at length in the Discussion, will serve as a useful guide for future research in this area.

- 1 Baxendale S, O'Sullivan J, Heaney D. Bright light therapy as an add on treatment for medically intractable epilepsy. *Epilepsy Behav* 2012; **24**: 359–64.

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Results for behavioural activation are overstated

The study by Moradveisi *et al.*¹ which is applicable to both secondary mental health and primary care, looks at the prospect of using minimally trained staff in delivering behavioural activation against pharmacological intervention in the treatment of severe depression. We would like to highlight the following points for further clarification.

First, an obvious problem of the study was the lack of a placebo arm, which would have lent credibility. As the cultural avoidance of antidepressants in Iran has been highlighted, adding a placebo group would have removed some bias such as paying for medication in the treatment as usual (TAU) group after 3 months and also in the analysis.

Second, sertraline was used at a suboptimal dose and was slowly titrated, against prevailing practice. A meta-analysis shows an optimum dose for sertraline between 100 and 150 mg/day – doses below the therapeutic range were significantly less effective, i.e. by 7%.² Sertraline reached its lowest therapeutic dose of 100 mg at 6 weeks. All drop-outs occurred before the mid-point assessment and only three were as a result of medication side-effects.

Third, there was a significant difference in the amount of attention that participants received in each group. Participants in the behavioural activation group received 50% more face-to-face sessions than the TAU group. The study did not adjust for this in the analysis.

Fourth, last observation carried forward (LOCF) was used in the study. However, 5% of drop-outs occurred in the behavioural activation group as opposed to a significant 30% from the TAU group. Last observation carried forward is used frequently in intention-to-treat studies but standard errors and confidence intervals from LOCF underestimate uncertainty.³ As there are no strategies for universal use, reasons for the choice of a certain method have to be provided when designing and analysing clinical trials.⁴ Last observation carried forward analysis seems to have favoured the behavioural activation group.

Many other limitations of the study are cited in the paper itself. Significant numbers of participants were recruited via advertisement or word of mouth, which seemed to have attracted more women and perhaps more psychologically minded individuals. It would have been helpful to include these advertisements as a supplement to the paper in order to identify any bias.

Finally, we wondered whether an ethics committee would allow this type of study to go ahead in the UK as it included individuals with severe depression. In England and Wales, before recruitment to a trial, potential participants must be assessed under the Mental Capacity Act 2005; in Scotland, the Adults with Incapacity (Scotland) Act 2000 (para. 72) must be used.⁵ Since the authors of the study state that 'the study's aim was to investigate whether a simple psychological treatment [...] would be a viable alternative to antidepressant medication [...] in a non-Western country', we are unsure of an equivalent law in Iran and whether this criterion was met.

- 1 Moradveisi L, Huibers MJH, Renner F, Arasteh M, Arntz A. Behavioural activation v. antidepressant medication for treating depression in Iran: randomised trial. *Br J Psychiatry* 2013; **202**: 204–11.
- 2 Bollini P, Pampallona S, Tibaldi G, Kupelnick B, Munizza C. Effectiveness of antidepressants. Meta-analysis of dose–effect relationships in randomised clinical trials. *Br J Psychiatry* 1999; **174**: 297–303.
- 3 Mallinckrodt C, Clark W, David S. Accounting for dropout bias using mixed-effects models. *J Biopharm Stat* 2001; **11**: 9–21.
- 4 Unnebrink K, Windeler J. Intention-to-treat: methods for dealing with missing values in clinical trials of progressively deteriorating diseases. *Stat Med* 2001; **20**: 3931–46.
- 5 General Medical Council. *Consent: Patients and Doctors Making Decisions Together*. GMC, 2008.

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Authors' reply: We thank Kripalani & Suleman for their critical remarks. Before addressing them point by point, a general remark is required. Our trial was an effectiveness, not an efficacy, trial. We compared a new treatment previously tested elsewhere (behavioural activation) with treatment as usual (TAU) (antidepressant medication) in Iran. An effectiveness trial aims to assess outcomes in usual care, not to test specific mechanisms, which affects the type of control condition(s). Some criticisms make sense from an efficacy study point of view, not from an effectiveness study point of view. Also of note is that the initial response to TAU was quite good, and that the longer-term response of behavioural activation accounted for its superiority.

We do not see how a placebo arm could have assessed cultural influences on TAU. To study this interesting topic, both a placebo and a natural course condition are needed to see whether placebo in Iran does worse than in other cultures compared with doing nothing.

Second, several sources state that 100 mg sertraline is a sufficient dose.^{1,2} Moreover, the dose is a valid representation of usual practice in Iran, as there is reluctance to increase the dose given findings that ‘often, adequate clinical activity, and saturation of the 5-HT transporters, are achieved at starting dosages. As a rule, higher dosages do not increase antidepressant efficacy, but may increase the risk of adverse effects’.²

Third, the difference in the amount of attention given is an inherent aspect of comparing behavioural activation and TAU in routine practice. Adjusting for this difference would lead to an invalid comparison in an effectiveness study. The question whether extra attention given to the TAU group would reduce the difference between behavioural activation and TAU is a legitimate one, but goes beyond the scope of this study.

Fourth, last observation carried forward was not used – this is a misinterpretation of the paper; intention-to-treat analysis was used, as it is the gold standard. Analysing only completers leads to biased conclusions. We used mixed regression analyses that use all available data and yield valid estimates under certain assumptions in the light of missing data.³ The suggestion is that a therapy-completers analysis would yield different conclusions. However, the effects are quite similar when only treatment-completers are analysed – Hamilton Rating Scale for Depression: time \times condition, $F(1,78.02) = 10.05$, $P = 0.002$; time squared \times condition, $F(1,78.40) = 7.94$, $P = 0.006$; Beck Depression Inventory: time \times condition, $F(1,78.02) = 6.84$, $P = 0.011$; time squared \times condition, $F(1,78.35) = 5.37$, $P = 0.023$.

Fifth, the influence of referral type was analysed, and tables with statistics are available online.⁴ It is difficult to understand that this was missed (e.g. ‘referral did not change the condition \times time and condition \times time squared effects’, p. 207). Moreover, if anything, the differences between conditions were stronger in participants who were referred by healthcare professionals.

Finally, all patients were capable of understanding the information about the offered treatments and making the necessary decisions. All individuals were seen by a psychiatrist to check eligibility, including capacity to consent to participate in the study, as part of the good clinical practice guidelines we applied.

- 1 Browne G, Steiner M, Roberts J, Gafni A, Byrne C, Dunn E, et al. Sertraline and/or interpersonal psychotherapy for patients with dysthymic disorder in primary care: 6-month comparison with longitudinal 2-year follow-up of effectiveness and costs. *J Affect Disord* 2002; **68**: 317–30.
- 2 Sadock BJ, Sadock VA. *Kaplan and Sadock's Synopsis of Psychiatry: Behavioral Sciences/Clinical Psychiatry* (10th edn): 1085. Lippincott Williams & Wilkins, 2007.
- 3 Schafer JL, Graham JW. Missing data: our view of the state of the art. *Psychol Methods* 2002; **7**: 147–77.
- 4 Moradveisi L, Huibers MJH, Renner F, Arasteh M, Arntz A. Behavioural activation v. antidepressant medication for treating depression in Iran: randomised trial. *Br J Psychiatry* 2013; **202**: 204–11.

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Effect of 9/11 on suicide: appropriateness of a time series model

Although the paper by Claassen *et al*¹ investigates an exciting issue, I have some concerns about the model identification. It seems that the authors identified the appropriate model of the time series only by using the Akaike Informations Criterion (AIC), which has certain limitations. For example, the selected ARMA (15,0) and ARMA (0,6) models are of high order and long memory. In general, the AIC suggests such models of high order only when a trend or seasonality is present in the analysed time series. Usually, if a time series is stationary, a model of an order below three is found.² A more complex method for model identification that avoids relying only on the AIC was introduced by Box & Jenkins.² Their algorithm includes several acquisition parameters in the process of model identification, which are:⁴ 0, make the series stationary, consider differencing; 1, choose a provisional model; 2, estimate the model parameters; and 3, check the adequacy of the model.

One key aspect is the requirement of stationarity. If the time series is not stationary, an ARIMA model should be considered instead of a mere ARMA model. The ARIMA model enables one to include terms for a trend or seasonality, respectively, directly in the model. The high order of the chosen model makes it likely that the time series in the paper indeed possesses a trend or seasonality. Furthermore, as the ultimate assessment of a correct model, Box & Jenkins demanded non-significant autocorrelations of the residuals, which were apparently also not checked in the paper. As these important aspects were not respected, the chosen model might not be correct.

Figure 1 below displays a time series with an underlying trend. When an ARMA model is assumed, the AIC suggests an ARMA (6,0), which does not fulfil the requirement of non-significant autocorrelations of the residuals on a significance level of $\alpha = 0.05$. Nevertheless, the simple differentiation of the time series leads to a straightforward ARIMA (1,1,2), which, in contrast to the previous case, meets this requirement.

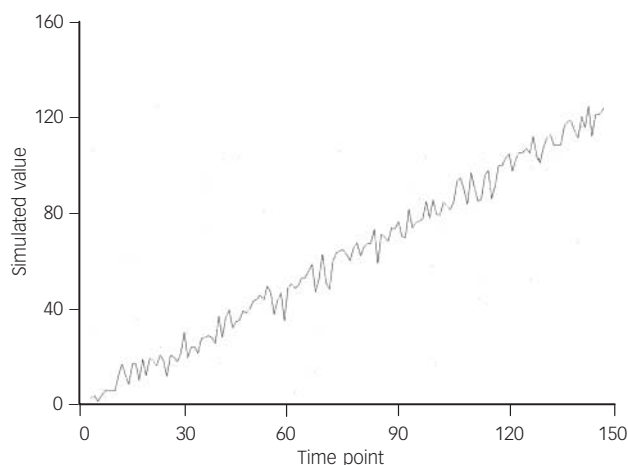


Fig. 1 Exemplary time series with an underlying trend.

The consequence of a non-fitting model would be a falsely estimated standard error, which would directly lead to insufficient statistical tests and thus incorrect P -values.^{2,4–6} When the control group of suicides in 1998 was regarded, an even larger post-9/11 effect over a period of 180 days was found than in the group of interest (suicides in 2001). This effect was rejected because of non-significant statistical tests, which is, as shown above, not

appropriate under the performed model identification. Therefore, it would be necessary to re-evaluate the considered time series in terms of model identification by the Box & Jenkins method and apply them again to the time series. I expect a notable change of results.

- 1 Claassen CA, Carmody T, Stewart SM, Bossarte RM, Larkin GL, Woodward WA, et al. Effect of 11 September 2001 terrorist attacks in the USA on suicide in areas surrounding the crash sites. *Br J Psychiatry* 2010; **196**: 359–64.
- 2 Rinne H, Specht K. *Zeitreihen: Statistische Modellierung, Schätzung und Prognose [Time Series: Statistical Modelling, Estimation, Prognosis]*. Vahlen, 2002.
- 3 Box GEP, Jenkins GM. *Time Series Analysis: Forecasting and Control*. Holden-Day, 1976.
- 4 Helfenstein U. Box–Jenkins modelling in medical research. *Stat Methods Med Res* 1996; **5**: 3–22.
- 5 Montgomery DC, Weatherby G. Modeling and forecasting time series using transfer function and intervention methods. *AIIE Transactions* 1980; **12**: 289–307.
- 6 Box GEP, Tiao GC. Intervention analysis with applications to economic and environmental problems. *J Am Stat Assoc* 1975; **70**: 70–9.

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Little evidence for the usefulness of violence risk assessment

Troquete and colleagues report a cluster randomised trial of the effect of violence risk assessment on future offending.¹ They found that people in the risk assessment group were non-significantly more likely to re-offend than those in the control group. We welcome this analysis of the practical value of risk assessment. There are now literally thousands of published violence risk assessment studies, most of which claim validity for their risk assessment method on the basis of statistical discrimination between violent and non-violent groups using measures such as the area under the curve (AUC) or other indicators of effect size.² Recent criticism of the AUC as an outcome measure has emerged because it does not reflect the accuracy of predictions in the real world, and even high AUC values are associated with a low positive predictive value (PPV) for rare events. However, the PPV of a risk assessment is only a proxy for the usefulness of a risk assessment. A risk assessment alone is not valuable unless it leads to reasonable interventions that can reduce future harm. Therefore, the utility of a risk assessment must ultimately be judged by its ability to contribute to harm reduction. In contrast to the large number of papers about the statistical aspects of risk assessment, there may be as few as four published controlled studies of the ability of risk assessment to reduce harm.²

The *British Journal of Psychiatry* has published two earlier studies of the utility of risk assessment. Abderhalden *et al* reported a cluster randomised trial of risk assessment among in-patients that found that intervention wards had a reduction in violence. However, interpretation of this study is difficult because the intervention wards had high rates of violence pre-trial and post-trial rates of violence in the experimental and control wards did not differ.³ Also in the *Journal*, van de Sande and colleagues reported a cluster randomised trial that found that risk assessment was associated with a reduction in violence but not seclusion among in-patients.⁴ In the nursing literature, Kling *et al* reported a study in in-patient settings that found that risk assessment was not helpful in reducing violence.⁵

Risk assessment has become the dominant paradigm in mental health practice, policy and legislation in most high-income countries. It should therefore trouble colleagues who support

‘evidence-based practice’ to know that there is so little evidence for the effectiveness of risk assessment.

- 1 Troquete NAC, van den Brink RHS, Beintema H, Mulder T, van Os TWDP, Schoevers RA, et al. Risk assessment and shared care planning in out-patient forensic psychiatry: cluster randomised controlled trial. *Br J Psychiatry* 2013; **202**: 365–71.
- 2 Wand T. Investigating the evidence for the effectiveness of risk assessment in mental health care. *Issues Ment Health Nurs* 2012; **33**: 2–7.
- 3 Abderhalden C, Needham I, Dassen T, Halfens R, Haug H-J, Fischer JE. Structured risk assessment and violence in acute psychiatric wards: randomised controlled trial. *Br J Psychiatry* 2008; **193**: 44–50.
- 4 van de Sande R, Nijman HLI, Noorthoorn EO, Wiersma AI, Hellendoorn E, van der Staak C, et al. Aggression and seclusion on acute psychiatric wards: effect of short-term risk assessment. *Br J Psychiatry* 2011; **199**: 473–8.
- 5 Kling RN, Yassi A, Smailes E, Lovato CY, Koehoorn M. Evaluation of a violence risk assessment system (the Alert System) for reducing violence in an acute hospital: a before and after study. *Int J Nurs Stud* 2011; **48**: 543–9.

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Authors’ reply: We agree with Wand & Large that there currently is very limited support for the use of structured risk assessment instruments as a method for violence prevention. So far only a small number of studies, four including our own, examined this issue. It is troubling that most research efforts seem to focus on the development of new risk assessment instruments and establishing their psychometric properties, rather than on testing the effectiveness of existing instruments. Although identification of predictors and development of instruments are crucial steps in the maturation of both risk assessment and forensic psychiatry, the field needs to move beyond these issues.

The most important risk and protective factors associated with recidivism have by now been established and are agreed on by the research community. There is no disputing the existence of correlations between mental illness, substance misuse, client well-being, quality of life and recidivism. That is why all, or a considerable selection of these factors, are commonly included in risk assessment instruments.^{1–3} It seems it is time to move forward and start investigating the benefits of risk assessment instruments and their contribution to more effective treatment interventions in terms of reduction of criminal and violent behaviour. As we ourselves have experienced, introducing randomised trials in clinical practice is difficult, but it can be done, and is an essential step before implementation can be advocated.

A definitive answer about the contribution of structured risk assessment to violence prevention cannot be given at this time. The first signs are not good. The four available studies find either no significant reduction of violent outcome, or the interpretation of their findings is problematic due to differences between study groups at baseline. Differences in clinical setting of the various studies further complicate the integration of findings. Our own data were collected in a community-based forensic mental health setting. In contrast, the other three studies were completed in acute psychiatric (admission) wards. These two settings service different populations, making comparisons less straightforward. It is too early for a proper systematic review on this subject, but the overall picture is not yet convincingly in favour of changing treatment policies by systematically employing structured risk assessment in clinical care.

On the other hand, our paper also shows that proper implementation in clinical care depends on personnel and organisational factors that need to be addressed in a coherent and persistent way before meaningful results can be obtained. The implementation of a randomised controlled trial has its particular challenges, but so does changing clinical practice in and of itself.⁴ As researchers, we may sometimes underestimate this gap between scientific evidence and the changes necessary in clinical practice for the implementation of evidence-based interventions. In order to reach the ultimate goal of prevention and to determine whether structured risk assessment may contribute, more studies are needed that assess the results of properly implemented and already established instruments in different forms of forensic care.

Declaration of interest

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- 1 Webster CD, Douglas KS, Eaves D, Hart S. *HC-20: Assessing the Risk of Violence. Version 2*. Simon Fraser University and Forensic Psychiatric Services Commission of British Columbia, 1997.
- 2 Nicholls TL, Brink J, Desmarais SL, Webster CD, Martin ML. The Short-Term Assessment of Risk and Treatability (START): a prospective validation study in a forensic psychiatric sample. In *Essential Writing in Violence Risk Assessment and Management* (eds H Bloom, CD Webster). Centre for Addictions and Mental Health, 2007.
- 3 de Vogel V, de Ruiter C, van Beek D, Mead G. Predictive validity of the SVR-20 and Static-99 in a Dutch sample of treated sex offenders. *Law Hum Behav* 2004; **28**: 235–51.
- 4 Forsner T, Hansson J, Brommels M, Wistedt AA, Forsell Y. Implementing clinical guidelines in psychiatry: a qualitative study of perceived facilitators and barriers. *BMC Psychiatry* 2010; **10**: 8.

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